Modern Concepts of Cardiovascular Disease

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THE HEMODYNAMIC CONSEQUENCES OF ATRIAL FIBRILLATION*

Atrial fibrillation in man is a condition which is characterized by undulatory and twitching movements of the atria. The lack of a normal coordinated contraction is based upon a disturbance of electrical impulse formation, the exact nature of which is obscure.[1] As a result, both electrical and mechanical evidences of atrial activity appear grossly disturbed. In addition, in most, but not in all cases, atrial fibrillation is associated with a total disorder of the ventricular rate and, in consequence, of pulse and pulse volume. This results in the well-known "irregular irregularity" of the heart beat which is almost the only way by which the diagnosis of atrial fibrillation can be made at the bedside; careful evaluation of the jugular venous pulse is another diagnostic aid. It is clear that the diagnosis based on the ventricular response is indirect and can only be carried to a high degree of suspicion. A definite diagnosis of the atrial "dysrhythmia" is only possible by graphic methods.

The ventricular irregularity is the result of multiple waves of excitation bombarding the atrioventricular junction which is known to be a region of limited capacity for conduction. The fastest ventricular response apparently coincides with the refractory period of atrioventricular nodal tissue and the longer intervals are multiples of this period.[2] On close analysis the "complete" irregularity, therefore, does follow a certain pattern, which is related to the conducting capacity of the atrioventricular node. More important, perhaps, is the observation that a reasonably slow, though irregular, resting ventricular rate may rise precipitously upon even mild exertion,[3-5] a fact that is not always overcome by digitalis therapy. Obviously, even with a normal minute volume this represents a needless expenditure of energy. At the onset of atrial fibrillation, the ventricular rate is often excessively high even at rest, and hemodynamic measurements in animals during this phase have demonstrated a decline in cardiac output.[6-8] This is an acute reaction which gradually corrects itself, and after some time cardiac output rises again to normal levels even though atrial fibrillation continues. In man, the sudden rise in heart rate at the onset of atrial fibrillation may explain the occurrence of heart failure in some patients shortly after the irregularity has occurred. In the following we are not concerned with these acute adjustments, but shall limit the discussion to "chronic" atrial fibrillation with a moderately slow resting ventricular rate.

If one considers the possible influences of atrial fibrillation on various hemodynamic parameters, two factors have to be considered: (1) ventricular irregularity and the tachycardia on exercise, and (2) the onset of what in effect might be called an atrial paralysis. If heart failure is the result of faulty energy conversion within the myocardium,[9] the sudden demand for increased energy expenditure resulting from a ventricular tachycardia, even if it only occurs on exertion, may overtax a diminished reserve. The rate-failure relationship may explain why occasionally heart failure or angina pectoris occurs in subjects with advanced cardiac disease when atrial fibrillation with a slow ventricular response is converted to a normal sinus mechanism which may have a basically faster and less controllable rate. We have seen such instances. On the other hand, heart failure may clear rapidly upon conversion to sinus rhythm even if the ventricular rate is changed but little and while the patient is confined to bed.[10] Subjective "improvement" upon conversion, even when congestive failure is not present, may be striking because the sudden cessation of a tumultuous ventricular action, if discernible to the patient, is often greeted with a great sense of relief. Yet some patients are able to separate this effect from what one might call "hemodynamic" symptoms; they may note ease of respiration with greater chest excursions (decrease in pulmonary turgescence?) and better exercise tolerance (improvement in cardiac output?), even if they have been unable previously to sense the irregular ventricular action.

The salient problem, namely, whether cardiac function improves upon conversion or not, centers around the question of how much the contraction of a normal or of an hypertrophied atrium contributes to diastolic ventricular filling. A recent textbook assigns supplementary diastolic filling by atrial contraction to "around 30

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per cent" of the total. This figure is based on open-chest experiments in dogs under vagal stimulation[11] and may not properly reflect the atrial contribution to ventricular filling under physiologic conditions and with an intact thoracic cage. There is, however, very little direct evidence bearing on this obviously crucial information, and there is none concerning the role of an hypertrophied atrium contracting against a narrowed orifice.

The following considerations may be of some help in this respect: Left heart catheterization by atrial puncture permits the almost simultaneous recording of pressure from the left ventricle and from the left atrium. Therefore, exact hydraulic principles may be applied to clinical problems with the hope of greater success than when such calculations were by necessity based on less exact measurements. Blood flow from the left atrium into the left ventricle through the fixed orifice of a severely stenosed mitral valve closely simulates conditions which apply to flow of any liquid across an orifice, providing the opening of this "nozzle" is at all times smaller than the chamber on either side.[12] Under such conditions, for each instance of time, the volume of diastolic filling is directly proportional to the square root of the pressure differences between the two reservoirs on either side of the orifice. If one plots the differences which may be obtained by left atrial puncture as the square root against diastolic filling time, the area under the curve is proportional to the volume of blood flow throughout diastole. In subjects with mitral stenosis and atrial fibrillation such a plot reveals high flow with rapid ventricular filling during early diastole, followed by a gradual decline until the onset of the next systole. If normal sinus rhythm is present, atrial contraction augments flow during the late diastolic phase, but to a surprisingly small degree. Such calculations indicate that an appreciable increase in flow during atrial systole is not likely to be encountered under any clinical condition. Perhaps of greater significance is the fact that in atrial fibrillation the rate of ventricular inflow at the end of diastole is still approximately 60 per cent or more of the peak value. One can imagine, therefore, how an increase in cardiac rate, with shortening of diastole, could seriously interfere with cardiac filling. These considerations demonstrate that impaired atrial contraction should interfere less with ventricular filling than an unbridled tachycardia. This applies to subjects with mitral valve stenosis, but may not apply under other circumstances.

Hemodynamic measurements in man obtained during atrial fibrillation and repeated after conversion to a normal sinus mechanism have always shown some increase in cardiac output, irrespective of the underlying etiology.[13-16] The average "improvement," however, has often been of questionable significance because of the rather large variabilities inherent in all flow measurements on human subjects, even when obtained by cardiac catheterization. In some instances improvement was striking; in others no effects could be demonstrated. Data from this laboratory have shown that in "compensated" subjects, output on mild exercise was often higher during normal sinus rhythm than during fibrillation even when cardiac status otherwise appeared relatively unchanged.[17] Repetitive studies of this sort performed on separate days (once before and once after conversion) are always subject to many influencing factors of which the presence or absence of atrial fibrillation is only one. The variables inherent in the design of such an experiment may be reduced if normal rhythm is restored during one experimental session, for example by the use of intravenous quinidine. Under these circumstances, however, the influence of quinidine itself has to be taken into account. Administered by the parenteral route, quinidine even in large doses exerts little if any hemodynamic effects, and certainly does not improve cardiac function (Table I). In subjects who converted to a normal sinus rhythm under such a regimen, no striking improvement in over-all cardiac function could be demonstrated. However, all patients who were successfully restored to a normal sinus rhythm responded with an increase in stroke volume on exercise, which is another way of saying that they had lost the exercise tachycardia so characteristic of many subjects with atrial fibrillation. Obviously a more economical situation prevailed. One might suspect that in some individuals this could spell the difference between normal function and heart failure. Table I shows some examples of such hemodynamic studies: Subject F.B. showed few, if any, alterations following conversion except those resulting from changes in heart rate; patient W.K., on the other hand, demonstrated obvious improvement of exercise performance after conversion, both in stroke and minute volume.

What might be the practical consequences of such considerations? Obviously there is no simple answer. The contribution that atrial systole makes to ventricular filling at rest and on exercise is still unknown, and in many subjects conversion to regular sinus rhythm is followed by little if any hemodynamic improvement. In some, cardiac output may rise slightly after conversion, or at least exercise tolerance may improve. In others in whom an excessive rise in the ventricular rate on exercise (or following vagal blocking) was present during the period of atrial fibrillation, the restoration of a normal sinus mechanism may be beneficial because now a more economical exercise response is achieved. Should one then insist on conversion once atrial fibrillation has been discovered? The answer here is a conditional yes, and partly of course because of factors not directly concerned with the hemodynamic consequences of the atrial disorder. If failure has occurred with the onset of the fibrillation and cannot be brought under control by the usual methods, either a lack of a needed atrial supplementary filling might be suspected as the cause, in spite of the arguments mentioned above, or an excessive rate response has occurred and conversion is indicated. Naturally, the well-known frequency of embolic episodes, particularly in rheumatic subjects of older age, has to be considered. Against these arguments for conversion one must also weigh the disadvantages of quinidine intoxication, the dislodging of recent thrombi with the onset of coordinate contractions, and the rather poor long-term success of maintaining a normal sinus

TABLE I

Examples of Hemodynamic Adjustments to Quinidine and to Atrial Fibrillation

Patient		Oxygen Consumption ml./min./m² STPD		Ventilation l/min. BTPS		Cardiac Output l/min./m²		Stroke Volume ml./beat/m²		Heart Rate beat/min.		Pulmonary Pressure mm.Hg		Systemic Artery Pressure mm.Hg	
		C.	Q.	C.	Q.	C.	Q.	C.	Q.	C.	Q.	C.	Q.	C.	Q.
1) R.C., NSR, Age 27															
0.8 Gm. quinidine, i.v.	R	161	147	8.4	8.0	5.7	5.7	74	59	70	88	24/10	18/5	140/76	132/72
(normal)	X	370	365	18.1	18.2	8.1	6.9	68	56	100	110	24/9	20/10	153/80	135/76
		AF	NSR	AF	NSR	AF	NSR	AF	NSR	AF	NSR	AF	NSR	AF	NSR
2) F.B., AF, Age 37															
1.2 Gm. quinidine, i.v.	R	130	122	4.7	4.0	3.4	2.5	74	56	84	56	18/6	22/15	100/60	115/70
(idiopathic)	X	518	542	20.8	23.1	5.0	5.4	67	95	135	95	24/12	28/15	150/85	150/70
3) W.K., AF, Age 58															
(arteriosclerotic)	R	109	104	6.7	7.6	1.6	1.3	41	52	75	50	25/0*	35/5*	125/80	140/80
	X	157	175	10.2	11.5	1.5	2.9	23	95	130	62	42/10*	47/7*	160/90	150/85

C.	Control
Q.	After quinidine
AF	Atrial fibrillation
NSR	Normal sinus rhythm
R	Resting values
X	Values on exercise
STPD	Corrected to 0°, 760 mm., dry
BTPS	Corrected to body temperature, barometric pressure, saturated
*	Right ventricular pressure

rhythm, particularly if large initial doses were required.

Many extenuating conditions have been mentioned that color the clinical picture of atrial fibrillation. Others have not been considered here. The decision to attempt to convert atrial fibrillation to normal sinus rhythm must remain an individual problem. The multiplicity of factors involved in that decision precludes the use of rules based on statistical evidence, and requires the discriminating ability of a careful physician.

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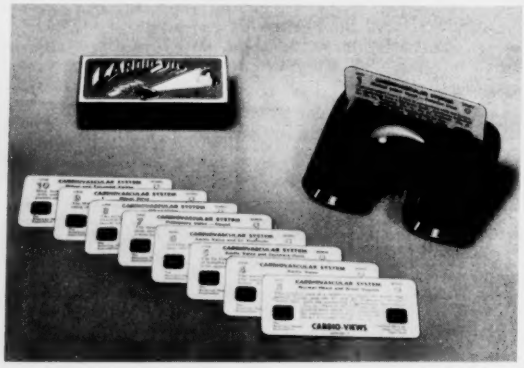

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